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JOURNAL ARTICLE

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Manidipine improves spermatogenesis in the stroke-prone spontaneously hypertensive rat

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We evaluated the protective effects of manidipine, which is a longlasting calcium-channel blocker, against damage to spermatogenesis arising from hypertensive vascular changes in stroke-prone spontaneously hypertensive rats (SHRSP). SHRSP showed severe hypertension at 11 weeks of age, followed by hypertensive changes in intratectional arterial of from 15 weeks of age. Manidipine Laward

intratesticular arterioles from 15 weeks of age. Manidipine lowered the blood pressure and the hypertensive vascular changes of intratesticular arterioles in SHRSP. The

the blood pressure and the hypertensive vascular changes of intratesticular arterioles in SHRSP. The percentages of atrophic seminiferous tubules and tubules with less-differentiated germ cells were increased in SHRSP at 23 weeks of age, although the administration of manidipine preserved spermatogenesis at a normal level. The transferrin concentration in testicular cytosol was comparable, whereas insulin-like growth factor-I (IGF-I) was reduced from 19 weeks of age in SHRSP. Manidipine preserved the normal IGF-I concentration. Therefore, manidipine prevented the development of hypertensive vascular changes in the testis and maintained normal Sertoli cell function. As a result, manidipine protected spermatogenesis in SHRSP. These findings also suggested that hypertensive vascular changes in the testes play the most important role in spermatogenic damage in SHRSP.

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